

# Cardiopulmonary Bypass in Patients With Pre-existing Coagulopathy

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**Abstract:** Patients with pre-existing coagulopathies who undergo surgical interventions are at increased risk for bleeding complications. This risk is especially true in cardiac surgical procedures with cardiopulmonary bypass (CPB) because of the necessity for heparinization and the use of the extracorporeal circuits, which have destructive effects on most of the blood components. In this review, cases of cardiac surgeries in patients with certain pre-existing coagulopathies are summarized, which could shed a light on future managements of such patients undergoing cardiac procedures with CPB. Pre-existing coagulopathies include antithrombin III deficiency, heparin-induced thrombocytopenia, cancer, factor XII deficiency, hemophilia, idiopathic

thrombocytopenic purpura, protein S deficiency, and drug-induced platelet inhibition. In summary, pre-existing coagulopathy in patients undergoing open-heart surgeries, if not recognized and appropriately managed, can cause serious complications. Management of patients undergoing cardiac procedures should include a routine coagulation work-up and a thorough past medical history examination. If any of the foregoing is abnormal, further evaluation is warranted. Proper diagnosis and management of the pre-existing coagulopathy disorders is of crucial importance to the surgical outcome and long-term morbidity. **Keywords:** cardiopulmonary bypass, coagulopathy, anticoagulation, heparinization, thrombosis. JECT. 2005;37:15-22

Patients with pre-existing coagulopathies undergoing surgical interventions are at increased risk for bleeding complications. This is especially true in cardiac surgical procedures with cardiopulmonary bypass (CPB) because of the necessity for heparinization and the use of the extracorporeal circuits, which have destructive effects on most of the blood components. In this review, cases of cardiac surgery in patients with certain pre-existing coagulopathies are summarized in Table 1, which could shed a light on future managements of such patients undergoing cardiac procedures with CPB.

## FACTOR XII DEFICIENCY

Factor XII is a component of the contact activation complex that contributes to initiation of the intrinsic pathway of coagulation. Despite a markedly prolonged activated partial thromboplastin time in the presence of severe Factor XII deficiency, these patients do not experience an increased risk of bleeding (1). However,

monitoring the heparin effect during cardiac surgery in patients with severe Factor XII deficiency is problematic because the usual tests of the intrinsic coagulation pathway require Factor XII to accurately reflect in vivo anticoagulation.

Several methods for managing anticoagulation in patients with Factor XII deficiency have been described. A simple technique is to administer empiric dosing of heparin on CPB without monitoring anticoagulation (2). Another approach is to obtain a baseline activated clotting time (ACT) and subsequently confirm the heparin effect by monitoring prolongation of the ACT (3,4). The third technique is to raise the Factor XII level preoperatively in these patients so the standard ACT monitoring of heparin therapy can be approached (5,6). In addition, blood heparin concentration could be measured directly during the perioperative period (7). For instance, Burman et al. (7) reported a successful case for the correction of atrial septal defect and patent ductus arteriosus on a 12-year-old girl with severe factor XII deficiency. Cardiopulmonary bypass lasted 48 min. Before bypass, 300 IU/kg heparin was given intravenously and neutralized after bypass by protamine sulphate. ACT was 620 seconds before heparin administration and more than 2000 seconds during bypass. Heparin concentration was maintained via heparin assay between 3.3 and 4.4 IU/mL during bypass, which was measured directly.

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**Table 1.** Cases of cardiac surgery in patients with certain pre-existing coagulopathies.

Coagulopathic Condition	Incidence in Cardiac Surgery Patients
Factor XII deficiency	1.5–3.0%
Hemophilia A	5–7%
Hemophilia B	1:30000
Idiopathic thrombocytopenic purpura	1–3.5:100000
Sickle cell deficiency	0.2%
Protein S deficiency	0.03–0.13%
Antithrombin III deficiency	1:2000–5000 (hereditary); 5–6% (acquired)
Heparin-induced thrombocytopenia	1–3%

More recently, Gerhardt et al. (1) presented a modified ACT test in patients with Factor XII deficiency. In this approach, a titration curve measuring the ACT with varying ratios of patient blood mixed with fresh-frozen plasma (FFP) was performed to identify assay conditions that would provide sufficient Factor XII activity from donor FFP to achieve normal baseline ACT. Modified ACTs were performed subsequently. This technique compensates for the absence of Factor XII and provides relatively accurate measures of heparin effect in patients with Factor XII deficiency. Limitations of this technique include requirement for exogenous Factor XII from donor FFP and a normal baseline modified ACT before heparin administration. In addition, differences between donor and patient plasma proteins and antithrombin III may affect the validity of the modified ACT.

## HEMOPHILIA

Hemophilia A and hemophilia B are sex-linked recessive inherited diseases affecting males only, with females acting as carriers. The conditions result in various degrees of Factor VIII or Factor IX deficiency, respectively (8). Nahas et al. (8) presented a case of successful coronary artery bypass operation on a mild hemophiliac. One hour before the cardiac catheterization, the patient received 2000 units of Factor VIII and half the initial dose was then given every 8 hours for a total of five doses. The evening before the procedure the patient received 4000 units of Factor VIII. The same dose was repeated just before surgery. This study demonstrated that direct myocardial revascularization is feasible in patients with hemophilia A if Factor VIII replacement is performed appropriately and conservatively.

In a review study that summarized the results of 12 cardiac surgical procedures performed in patients with hemophilia at a single center from 1979 to 1998, the authors concluded that both major and minor cardiac procedures could be performed safely in patients with hemophilia (9). Some guidelines to facilitate future management of such patients also were proposed:

1. Team approach.
2. Factor replacement: a bolus of factor concentrate is administered preoperatively to achieve a target level of 100%; a repeat bolus of factor concentrate is administered post bypass, prior to wound closure and commencement of a factor infusion; in patients with Hemophilia A, ongoing factor replacement is achieved by continuous intravenous infusion.
3. Standard heparinization during cardiac bypass and reversal with protamine sulphate appear to be safe.
4. Monitoring: factor assays in addition to routine coagulation assays should be performed daily in the postoperative period.

In addition, desmopressin acetate (DDAVP) also has been reported to be given perioperatively to some patients with hemophilia undergoing cardiac procedures. The administration of DDAVP results in a dose-dependent predictable increase of all Factor VIII-related activities, and its use is recommended in mild forms of hemophilia (10,11). DDAVP causes the release of factor VIII from endothelial cells to which Factor VIII has been bound. It is not effective in severe cases since little to no Factor VIII bound to endothelial cells. Because DDAVP also stimulates the release of plasminogen activators, an antifibrinolytic agent is administered simultaneously.

## IDIOPATHIC THROMBOCYTOPENIC PURPURA (ITP)

ITP is an autoimmune condition arising from antibody binding to the glycoprotein IIb/IIIa complex on the platelet membrane. Antibody-coated platelets are prematurely removed from the circulation by attachment to macrophage Fc receptors, followed by phagocytosis. Traditionally, chronic ITP is defined as the persistence of thrombocytopenia for more than 6 months (12).

The increased complication rate of postoperative bleeding in patients with ITP who undergo cardiac operation is attributed to thrombocytopenia and impaired function of circulating thrombocytes as well as both the deleterious effects of cardiopulmonary bypass on platelets and the necessity for full anticoagulation (13).

Several forms of therapy have been used for ITP including prednisone, splenectomy, danazol, and variety of immunosuppressants (14). Prednisone is usually the initial treatment in ITP. If the patient fails to respond to steroids, cytotoxic drugs are administered. Splenectomy is usually considered an alternative treatment for patients with ITP. High-dose intravenous immunoglobulin has been recommended as emergency treatment. (14). Plasmapheresis has been successfully applied to remove autoantibodies in the treatment of severe forms of various autoimmune diseases (12).

In a case performed on a 59-year-old man with a 1-year history of ITP (12), coronary artery bypass graft operation was performed successfully using very fresh whole blood, steroids and intravenous immunoglobulin. In addition to oral prednisolone therapy, the patient received five consecutive dose of immunoglobulin. Further more, Aprotinin was given intraoperatively according to the Full-Hammersmith protocol (15).

Similarly, Nakamura et al. (15) reported that prednisone and the immunoglobulin treatments were administered preoperatively for a patient who had valvular disease with ITP. Platelet transfusion and nafamostat mesylate, a broad-spectrum inhibitor of inflammatory response, were used during the operation. The double valve replacement and tricuspid annuloplasty were performed successfully.

In addition, Christiansen et al. (16) showed that administration of blood products, especially platelet concentrates, post operatively represents a useful step to enhance platelet counts in patients with ITP. It is suggested platelet concentrated are transfused empirically, if platelet counts fall to less than 60/ng in these patients (17).

## SICKLE CELL DISEASE

Patients with sickle cell disease undergoing CPB require a reduction of hemoglobin S fraction to less than 30% to avoid a sickling crisis potentially induced by hypothermia, hypoxia, or hypotension (18–21). Hemoglobin S reduction can be accomplished by preoperative outpatient therapeutic phlebotomy, exchange transfusion, and an acute whole blood volume exchange transfusion before CPB.

In a successful mitral valve repair surgery with CPB performed on a 17-year-old male with severe sickle cell disease, blood component sequestration and red blood cell exchange were used to enhance reduction of the hemoglobin S mass (18). Sequestration before CPB decreased the patient's hemoglobin S concentration from 79% to 67%. Upon initiation of CPB, the hemoglobin S concentration was further reduced to 21% by an exchange transfusion with the CPB circuit. Hemoglobin S was replaced with Hemoglobin A to protect against a sickling crisis. Autologous plasma was provided to maintain volume and preserve coagulation factors.

However, Frimpong-Boateng and colleagues (22) reported the successful management of two children aged 12 years with sickle cell anemia, several sickle cell crises, and multiple blood transfusions who underwent CPB for mitral valve replacement. No preoperative or intraoperative exchange transfusions were used in the two cases. Furthermore, moderate hypothermia in both cases was not associated with adverse sequelae. Meticulous care was taken to avoid dehydration, hypoxia and acidosis during pump bypass.

## PROTEIN S DEFICIENCY

Protein S is a vitamin K-dependent plasma protein integral to the maintenance of normal function of the coagulation system (23). It is a cofactor of protein C, a strong inhibitor of activated coagulation Factors V and VIII. Activated protein C also increases the level of tissue plasminogen activator by increasing its release from the endothelium and by inhibiting its inhibitor (24). As a result, protein S and protein C act as agents of fibrinolysis and anticoagulation, that may, result in spontaneous thrombosis when levels are deficient or abnormal (25).

Protein S deficiency is inherited in an autosomal-dominant fashion (26). In Protein S-deficient patients, some recommendations have been made concerning anticoagulation (26). Short-term prophylaxis with anticoagulant was administered to patients in high-risk situations (surgery, immobility, and pregnancy). If deep venous thrombosis or pulmonary embolism was present in any patient, long-term anticoagulation is started. The transfusion of fresh frozen plasma units before surgery may have some theoretical benefit to enhance the level of protein S in these patients (25–27).

It has been reported that a patient with severe hereditary protein S deficiency underwent successful coronary artery bypass grafting without incidence of thromboembolism (25). Intraoperatively, the patient was given heparin to maintain an ACT of greater than 480 seconds while on CPB. Protamine was administered in the usual fashion after cessation of CPB to reverse the heparin effect.

However, in a case study on a 71-year-old man with Protein S deficiency undergoing coronary artery bypass graft surgery, Villacorta et al. (27) showed that 30 minutes after the usually dose of protamine was administered, two vein grafts were occluded. CPB was reinstated and new anastomoses were performed. The protamine was administered only in half-dose. Heparin was administered to maintain an activated coagulation time of 2.5 times normal subsequently. It was then recommended that the use of lower dose of protamine to be successful to avoid the intraoperative thrombosis of the vein grafts in patients with Protein S deficiency undergoing cardiac surgeries with CPB.

## PLATELET GLYCOPROTEIN (GP)IIb/IIIa INHIBITION

Although the use of the potent inhibitors of platelet glycoprotein IIb/IIIa is a major breakthrough in the treatment of acute coronary syndromes (28), operating on patients with active GP IIb/IIIa inhibition represents a major challenge for the surgical team (29,30).

A commonly used active GP IIb/IIIa inhibitor is Abciximab. Abciximab is a murine-derived monoclonal anti-

body with a large molecular size that acts through steric hindrance of the receptors. Abciximab has a high platelet affinity and remains bound to circulating platelets for up to 15 days after infusion until cleared by the reticuloendothelial system. No dosage adjustments are necessary for renal or hepatic dysfunction (31,32). Abciximab has been associated with a moderate increase in all bleeding complication associated with percutaneous intervention (31). Bleeding complications with abciximab in cardiac surgical patients varied in clinical trials and in individual series (32). Several series reported significant increase in blood loss, chest tube drainage, and transfusion needs (32). The existing bleeding seen in abciximab-treated patients has raised concern over the potential for increased risk of postoperative cardiac tamponade and resultant morbidity.

To minimize the real and perceived bleeding effects of abciximab, several components for the surgical management of patients who has been receiving platelet inhibitors have been described including transfusion therapy, reduced heparinization, and delay of surgical intervention when possible (33). It is suggested that the interval from cessation of abciximab administration to operation is critical in determining the degree of coagulopathy after the operation (33). Patients who underwent an emergency operation within 12 hours of administration had significantly greater transfusion requirements and bleeding complications. However, delay must be considered within the context of the stability of the patient and the need of reestablish blood flow with definitive therapy.

The ACT is prolonged in abciximab-treated patients, but the implications for clinical anticoagulation during surgical revascularization are unclear. Initial reports of excessive bleeding complications with empiric heparinization in abciximab-treated patients during CPB have led to reduced heparin anticoagulation, with reduction in bleeding risk (34,35). However, reduced heparin dosage may place the patient at risk for prothrombotic events during CPB. Current recommendations are empirical, based on time since abciximab administration. With recent administration (<12 hours), it has been recommended that the initial heparin bolus be a half dose before CPB, with additional heparin to achieve a target ACT of between 400 and 500 seconds (36). For delayed operation (>12 hours), standard full-dose heparin should be given, with titration for a target ACT of 400 to 500 seconds (37). It has been argued that full-dose scheme, regardless of ACT or abciximab interval, is the most conservative regimen to avoid under anticoagulation.

No current method of reversal exists for GP IIb/IIIa agents. Manufacturer guidelines suggest platelet transfusion to bind free drug in the plasma (32). Transfusion therapy with pooled platelets is essential to increase the number of functional platelets. It is also recommended

that the use of aprotinin as an adjunct to homeostasis in patients requiring an emergency operation in the face of GP IIb/IIIa inhibition. The use of hemoconcentrator during cardiopulmonary bypass in an ex vivo perfusion model has been described to reduce plasma levels of abciximab (38), allowing transfused platelets to remain functional instead of absorbing available abciximab. This method of abciximab elimination could reduce platelet transfusion in the cardiac surgical patients.

### ANTITHROMBIN III (AT III) DEFICIENCY

AT III deficiency can be either hereditary or acquired (39). Hereditary AT III deficiency is uncommon. Acquired AT III deficiency may result from reduced synthesis, increased AT III turnover, or increased excretion. AT III has significant inhibitory action against many serine proteins in the coagulation and fibrinolytic systems (40).

Patients undergoing cardiac surgery who have been receiving preoperative intravenous infusion of heparin may have an acquired AT III deficiency, which lead to an inadequate systemic anticoagulation during CPB and to the phenomena of consumptive coagulopathy due to excessive thrombin activation (41).

Patients with AT III deficiency can be treated with whole blood, FFP, or pooled human plasma, AT III (42). AT III preparations could withstand heat treatment without significant loss of biologic activity (43), which allows the application of a viral inactivation step. No transmission of the acquired immune deficiency syndrome, hepatitis B, or non-A, non-B Hepatitis, via AT III has been reported (44).

In a case study presented by Van Norman et al., AT III activity levels and the respective activated coagulation time (ACT) were studied before, during and after CPB with the administration of exogenous AT III was reported (39) on a patient who had received intravenous heparin and underwent coronary artery bypass surgery. It was found that the AT III activity level was about half of normal in this patient at baseline and the baseline ACT was 225 seconds. After heparin was given at a dose of 500 U/kg, the resultant ACT was 347 seconds. Exogenous AT III raised the ACT to more than 600 seconds without additional heparin administration.

Rossi and colleagues (45) evaluated the effectiveness of intraoperative administration of AT III to improve anticoagulation and preserve the hemostatic mechanisms during CPB in patients with unstable angina under heparin treatment. The 22 patients were divided into two groups; one group received AT III plus heparin before aortic cannulation and the other group received only heparin. It was found that the group that received AT III had fewer transfusions, less chest-tube drainage, and lower level of throm-

bin-antithrombin complex than the group that did not receive AT III. It was concluded that intraoperative administration of AT III concentrates allowed adequate anticoagulation during CPB and attenuated the coagulative cascade activation and the consequent consumptive coagulopathy.

## **HEPARIN-INDUCED THROMBOCYTOPENIA (HIT)**

HIT is defined as a decrease in platelet count induced by the administration of heparin for therapeutic purposes (46). Mild thrombocytopenia is the result of a direct agglutination effect of heparin on platelets, which is not associated with thrombosis and resolves within a couple of days (46). The immune form of heparin induced thrombocytopenia, which is associated with both arterial and venous thrombosis, is serious and life threatening (47). This response is the result of antibodies formed against the platelet factor 4 (PF4)/heparin complex. The platelet-aggregating factor in HIT sera has repeatedly been shown to be IgG (48). Activation of platelets results in their lysis and agglutination. PF4 can also bind to heparin-like molecules on the endothelial surface to provide targets for antibody binding and local injury (49).

In patients with acute HIT who require heart operations, several options can be chosen. One option is to wait for the HIT antibodies to become undetectable and then to operate using a brief intraoperative exposure to heparin. The theory underline this option is that during secondary immune responses the concentration of antibody in the serum begins to increase no earlier than three days after the challenge (50). Moreover, in the absence of heparin, HIT antibodies are not thrombogenic. Thus, it has been postulated that in patients with a history of heparin-induced thrombocytopenia who are challenged with heparin, the HIT antibody concentrations should start to increase only after the heparin is completely cleared from the circulation.

In a study performed by Potzsch and Klovekorn (51), on 10 patients with a history of acute HIT who required CPB, heparin was used. During the acute episode, all patients had detectable HIT antibodies on the heparin-induced platelet-aggregation assay. At the time of surgery, all patients were negative for these antibodies. The CPB were performed with the use of heparin without any problems. None of the 10 patients had prolonged thrombocytopenia. It was concluded that antibody-negative patients with a history of heparin-induced thrombocytopenia who undergo CPB should be treated according to established heparin protocols. The use of heparin should be restricted to the operative period. Postoperative anticoagulation should be achieved with alternative anticoagulants (51).

However, this approach is not suitable for an unstable patient because it might take several months for antibody levels to decline sufficiently. A second option is to perform heart operations using heparin substitutes.

Low molecular weight heparins have been documented as an alternative of unfractionated heparin on patients with HIT for CPB (52). In 1983, it was reported the first successful use of nadroparin for CPB in a 66-year-old man with HIT undergoing an emergency pulmonary thrombectomy (53). Later, in a case report, enoxaparin was used as for anticoagulation during CPB on a patient with HIT undergoing heart transplantation (54). In addition, argatroban (55), danaparoid (56), ancrod (57), and r-hirudin (58) all have been reported to substitute unfractionated heparin during cardiac procedures on patients with HIT.

Although the aforementioned cases have shown that several heparin alternatives can be used as anticoagulants during cardiac surgeries, all of these agents have their significant limitations. The cross-reactivity of HIT antibodies with a low molecular weight heparin may be found in approximately 90% of patients with HIT (59), making low molecular weight heparins a potentially dangerous alternative to unfractionated heparin in these patients. In addition, no current coagulation assay or set of assays has been shown to indicate whether satisfactory CPB anticoagulation has been achieved with any low molecular weight heparin. Danaparoid does not affect the ACT or aPTT, requiring antifactor Xa determination. With lepirudin, the aPTT may be used to monitor patients who receive small dose (60). However, the levels of r-hirudin recommended for anticoagulation during CPB exceed those that can be effectively monitored with either the aPTT or ACT (61). The ecarin clotting time assay, a clot-based method that uses a prothrombin-activating snake venom derivative and can be measured with TAS instrument (Cardiovascular Diagnostics, Raleigh, NC) produced adequate dose-response curves with r-hirudin in patients during CPB (61) but is not widely available. In addition, ecarin clotting time can be substantially prolonged by hemodilution or CPB related reductions in procoagulant proteins. More over, the lack of antidotes for these agents means that severe postoperative bleeding can occur (62).

Recently, a new alternative, bivalirudin, has been reported successfully used in cardiac surgery patients with acute or previous HIT for anticoagulation during CPB (63–65). Like argatroban, and hirudin, bivalirudin is a direct thrombin inhibitor, binding directly and specifically to thrombin and do not require antithrombin, heparin cofactor II, or any other cofactor for their effect (66). Bivalirudin is a reversible thrombin inhibitor, has a rapid onset, a short half-life of approximately 25 minutes and a rapid plasma clearance of about 3.4 mL/minutes via a combina-

tion of renal and proteolytic mechanisms. It has no reversal agent but can be removed by hemodialysis (66). Hemofiltration can remove as much as 70% of bivalirudin from treated blood. ECT is recommended for intraoperative monitoring although the activity of bivalirudin has been reported successfully monitored with ACTs (63).

## CANCER

Cancer patients who undergo major surgeries are at significantly higher risks of thromboembolic and bleeding complications. It has been shown that these patients are twice as likely to have thromboembolic or bleeding postoperative complications and are three times as likely to die of pulmonary embolism compared to non-cancer patients (67,68). Therefore, preventative prophylaxis of thrombosis and bleeding is an essential component of surgical care of the cancer patients.

The pathophysiology of the prothrombotic state in the cancer patient is dependent upon the type of tumor, extent of disease, host response to the malignancy, and specific therapies (69). In patients with solid tumors, a low-grade activated coagulation can result in arterial or venous thrombosis (70). A number of tumors, such as gastric and pancreatic tumors, have been shown to express tissue factor-like activity, which mediates excessive thrombin generation (71). In certain tumors a cysteine protease procoagulant capable of activating factor X has been described (72). Tumor infiltrating macrophages, an important mediator of the anti-tumor response, have been shown to express tissue factor activity (73) and a number of cytokines, which are capable of inducing endothelial tissue factor activity and down-regulating endothelial anticoagulant activity (74). Inflammatory cytokines stimulate increased synthesis and release of acute phase plasma proteins, such as fibrinogen, factor VIIa (75) and factor VIII, which further enhance the procoagulant hemostatic environment (74). In addition, the hypercoagulability that occurs with cancer has been described to associate with the decreased levels of anticoagulant factors, e.g., antithrombin or the proteins of protein C pathway (76).

Moreover, the coagulation disorder may be explained, in part, by the additional thrombotic risk incurred by the therapeutic interventions used in the treatment of the cancer patient, such as surgery and chemotherapy (69). Chemotherapy administration is associated with thrombosis due to direct endothelial cell injury or to a variety of speculative mechanisms, which include clotting activation or other hemostatic effects, vasculitis, and vasospasm (77).

In contrast to the cancer-associated thrombotic coagulopathy that is chronic, the hemorrhagic coagulopathies are more fulminant and fatal. The mechanism of acute disseminated intravascular coagulation (DIC) in most instances is not known, but is thought to be excessive throm-

bin production, which leads to increased consumption of platelets, coagulation factors, and inhibitors of coagulation. There may be primary or secondary fibrinolysis. In acute promyelocytic leukemia, release of progranulocytes from the degranulation of promyelocytes is one mechanism triggering DIC. The DIC is exacerbated by tumor cell lysis caused by the administration of chemotherapy (70).

Despite the significantly increased risk of thromboembolic and bleeding complications in cancer patients, a number of cases of patients with cancer who successfully underwent operations with CPB have been documented (78–81). ACT is commonly used as the index of adequacy of anticoagulation during perioperative period, whereas heparin is used as anticoagulant for CPB.

Back in 1982, Chun et al. (82) presented a case report that an aortic valve replacement performed on a 46-year-old male with chronic myelogenous leukemia. Undergoing moderate hypothermia and CPB, the patient underwent replacement of a prolapsed aortic valve with a no. 29 Bjork-Shiley aortic prosthesis, and the ascending aorta was replaced with a 33-mm woven Teflon tubular prosthesis. CPB time was 2 hours and 50 minutes, and aortic cross clamp time was 1 hour and 54 minutes. There were no complications at the 5-month postoperative follow-up.

Stewart et al. (81) reported that cavoatrial tumor thrombectomy were performed using CPB on 8 patients. Heparin was administered as anticoagulant and cannulation performed with superior vena caval drainage and ascending aortic return. Patients were cooled to 30° to 32°C. The cardiectomy suction was used to aspirate hepatic venous and coronary sinus return, and the tumors were carefully peeled from the vena cava and right atrium. Cardiopulmonary bypass was weaned and terminated after the patient was warmed to 37°C.

In summary, pre-existing coagulopathy in patients undergoing open-heart surgeries, if not recognized and appropriately managed, can cause serious complications. Management of patients undergoing cardiac procedures should include a routine coagulation work-up and a thorough past medical history examination. If any of the foregoing is abnormal, further evaluation is warranted. Proper diagnosis and management of the pre-existing coagulopathy disorders is of crucial importance to the surgical outcome and long-term morbidity.

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